Lead poisoning in Hawaii: 1990

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Although lead (Pb) is one of the oldest known and most thoroughly described toxins, it continues to be a significant health hazard in 1990. There has been much progress in defining the nature and extent of low-level lead toxicity during the past decade. There continues to be insidious sources of lead toxicity in our environment, in water, food, paint and contaminated soil. As the epidemiology of lead poisoning is more clearly defined, toxicities are recognized as the result of lower and lower levels of exposure. Recognition of low-level lead exposure and the primary prevention of its effects on health requires a keen awareness of high-risk environments as well as the subtle symptoms and signs of lead poisoning. A high index of suspicion by primary care physicians plus government support are necessary to implement successful prevention programs.

Historical perspectives

Lead is probably the oldest neurotoxin known to man. It has been obtained and used by man since the earliest recorded times[1]. The uses for lead throughout the ages have been as variable as the mind can imagine. As a medication it has been used for everything from contraception to management of epilepsy[2]. Latin American and Southeast Asian cultures, even today, ingest lead oxide as a remedy for fever, rash, and digestive complaints[3]. The ancient Roman practice of sweetening poor vintage wines with lead-containing mixtures has been in vogue as late as the 18th century in the United States. The use of lead-containing substances as additives in paints, cosmetics, and ceramic glazes have also been well recognized as a cause of toxicity throughout the ages.

The toxic effects of lead were known most likely to both the Greeks and Romans. Hippocrates described a severe attack of abdominal pain (presumably "lead colic") in a man who was extracting metals. Nicander, in the second century BC, noted an association between exposure to lead and pallor, constipation, colic, and paralysis. Pliny (AD 79) served warning to the Romans of the danger of inhaling fumes produced by the smelting of lead. In 1763, Benjamin Franklin warned of lead toxicity from drinking rum distilled in lead-condensing coils, causing colic and wrist drop. Sir George Baker in 1767, traced the disease described as "Devonshire colic" to cider prepared in lead presses. In 1839, Tanquerel des Planche published a famous study of 1200 cases of lead poisoning[4].

The use of lead in industry has been a cause of environmental pollution in every industrialized nation. Analysis of sequential layers of ice in Greenland have documented a twenty-fold increase in lead pollution over the past 200 years[5]. The current world production of lead is approximately 2.5-million tons per year with 40% of it consumed annually by industries in the United States.

Lead in Hawaii

Surveillance of Hawaii's children for potential lead toxicity has been attempted on several occasions over the past 20 years. In 1972, blood samples were taken from 156 Head Start children in Honolulu for determination of free erythrocyte protoporphyrin (FEP). The results showed no evidence of toxicity; however, there was a question as to the reliability of the laboratory performing the tests[6].

In 1973, blood lead-levels were performed on 76 children from Kalihi valley as a part of a comparative study featuring Honolulu, Hawaii, and Newark, New Jersey[7]. The mean blood lead-level in this series of Honolulu children was 17 µg/dl with a range of 10 to 30 µg/dl; 90% of the children tested had blood lead-levels less than 20 µg/dl. Seventy-six age- and sex-matched preschool children from Newark were compared to the Honolulu cohort and found to have a mean blood lead-level of 28 µg/dl with a range of 11 to 60 µg/dl; 26% of children tested had levels above 30 µg/dl. Based on this study, it was assumed that Honolulu was relatively free of childhood lead-poisoning when compared with the older, more polluted city of Newark.

In 1974, the State of Hawaii Department of Health (DoH), Maternal and Child Health Branch, performed a pilot survey to determine the need for more widespread screening for lead-poisoning in Hawaii. One hundred and ninety-eight children under 6-years of age were selected from a variety of urban and rural areas on Oahu. Children were chosen for screening that

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had lived most or all of their lives in older housing. Three separate local and Mainland laboratories were utilized in order to check on lab variations. Results showed that 2 children with lead levels of 40 to 44 µg/dl, and 11 children with levels of 30 to 39 µg/dl. This pilot screening project again validated the impression that Hawaii had no major lead toxicity problem, although it was recommended that an increased awareness of the possibility of lead-intoxication in children in high-risk areas be posted.

In 1986, DoH investigators screened children at 10 preschools in Honolulu for excessive exposure to lead in order to determine the extent to which children attending preschools near major roadways in Honolulu might have been exposed to lead in the soil, the result of deposition from automobile exhaust emissions (Unpublished data). The schools selected were on the basis of the level of lead in the soil. The children's medical records at major medical centers in Honolulu were scrutinized.

Altogether, 341 (43.3%) of the 788 children enrolled at these schools were screened for evidence of undue exposure to lead; of these, 7.9% had FEP levels of ≥ 35 µg/dl and required follow-up blood lead testing. None of these children were unduly exposed to lead; that is, blood lead levels were below 25 µg/dl. Soil lead levels at the schools sampled ranged from an average of 32 parts per million (ppm) to 461 ppm, comparable to levels found in other urban centers in the United States.

These results are consistent with previous studies of a preschool located immediately adjacent to, and normally downwind of, an H-1 freeway viaduct in Manoa where, in June, 1983, 21 (52.5%) of the 40 children enrolled at the school were screened for evidence of excessive lead exposure by FEP measurements. Seven (33%) of the 21 children examined had FEP levels > 35 µg/dl. Follow-up blood lead screening indicated none were unduly exposed to lead. On December 18, 1984, 25 (52.5%) of the children enrolled at the school were re-examined; only 2 (8%) of the 25 children examined had FEP levels of ≥ 35 µg/dl; even so, both had blood lead-levels < 50 µg/dl.

These studies indicate that the prevalence of unacceptable lead exposure in children in Honolulu is well below the national average. Recent data suggest that about 6 to 7% of children tested in cities by blood-lead, screening programs met the criteria for excessive absorptions of lead (NAS, 1980). Indeed, the low incidence of lead-poisoning in Honolulu is surprising, considering that lead-based paints have been used extensively in Hawaii, and that soil lead-levels were similar to that in cities in the mainland United States, where automobile emissions contribute significantly to soil lead levels (Quarles, et al., 1974; EPA, 1977; Mielke, 1983).

In 1987, an unusual problem was identified in the South Kona area of the Island of Hawaii, where emissions of sulfur dioxide from the Kilauea Volcano Rift Zone had created an "acid-rain condition". Although the acid water per se did not pose a significant health risk, it had the potential to leach lead and other metals out of plumbing systems. This problem was brought to the attention of public health officials because of complaints of "blond hair turning green" from the copper in household plumbing systems.

Initial sampling of catchment water from 5 homes in South Kona found pH levels ranging from 4.08 to 6.25; 2 homes had lead in catchment water above the current Maximum Contaminant Level (MCL) of 50 parts per billion (ppb). In July 1988, DoH investigators systematically sampled 75 systems representative of all regions of the Island of Hawaii for lead and pH. While investigators found only a minor correlation between lead levels in water and pH, 28 percent (21/75) of the systems sampled had lead-levels above the proposed EPA standard for lead in public water systems, which is 20 ppb.

To ensure that the public was not being unduly exposed to lead-containing catchment drinking water, the DoH offered all residents who used catchment water for drinking, to have blood tests as well as their water tested for lead. Approximately 3,000 people were screened for lead exposure using the zinc erythrocyte protoporphyrin (ZPP) method; about 5% had elevated ZPP levels. None of those with elevated ZPP levels was found to be unduly exposed to lead from drinking lead-contaminated water (ZPP can be elevated as a result of other disorders, such as iron deficiency). In addition, over 2,000 water samples were analyzed; lead concentrations ranged from less than detectable limits (20 ppb) to levels as high as 7000 ppb. Preliminary results showed a correlation between water-catchment lead content and blood lead-levels (unpublished results).

Follow-up investigations have found that lead-based paint used in roofing and as a sealant for water tanks, lead flashing, lead-headed nails, and lead solder used in gutters and piping could have contributed to high lead-levels in catchment water systems. Since samples from catchment systems thought to be free of leaded materials as well as the systems with leaded materials in areas that did not have acidic rainwater both showed traces of lead, the DoH has recommended that homeowners replace leaded materials in their home catchment systems and test all catchment water before using it for drinking or cooking.

To determine the incidence of recognized lead poisoning, medical records at the major medical centers in Honolulu were reviewed for the 16-year period 1970 through 1985. Only two cases of lead poisoning among children were identified; both very likely were exposed to lead-based paint. One case was a 4-year-old boy living in Waianae. He was admitted to the hospital for workup of nocturia and flank pain. Blood lead-levels of 39 µg/dl were noted in his record. Parents of the child had indicated he ate paint at his home. The second case was a 4-year-old boy living in Honolulu who was admitted for anemia. Blood tests revealed blood lead levels of 77 µg/dl and urine lead levels of 79 µg/dl. This child was known to have chewed on a stairway railing. Testing paint for lead in both instances was not possible.

The only other case of lead-poisoning found in the medical records review was that of a 54-year-old woman living in Honolulu who was hospitalized for anemia, abdominal pain, and vague neurological complaints. The source of lead was not determined in her case. Her dietary history included the consumption of a large amount of Chinese herbs which may have been prepared using lead utensils, but the testing of the

herbs and various utensils for lead apparently was not done.

The results of these studies support the conclusion that children in Honolulu are not at elevated risk for lead exposure. However, one must be cautious in extrapolating these results to rural areas where one might expect older housing with deteriorating lead-based paint and, possibly, other sources of lead.

The sources of lead

Tables I to III include a partial list of potential sources of lead which may be helpful to the primary-care physician seeking a history of lead exposure from his patient. Since Hawaii is free of major industrial sources of lead, household and home environment exposures to lead are more likely to be the source for lead poisoning in children. Paint used in homes built prior to 1950 often contained large amounts of lead carbonate. Old, peeling, lead-based paint, or dust from demolition of old buildings, provide a tasty sweet treat for the toddler exploring the environment. Home renovation projects are still a source of lead poisoning[8]. Paint chips, fumes, or dust, as houses in inner city areas are renovated, provide a major source of childhood lead-poisoning. Since 1977, household paint is not allowed to contain more than 0.06% lead by dry weight.

TABLE I

SOURCES OF EXPOSURE TO LEAD IN HOUSEHOLD

Lead-based paint
Lead-glazed pottery
Antique pewter cookery
Lead toys, shot, bullets
Fishing weights
Foil on wine bottles
Hair dyes, cosmetics
Food, drinking water
Contaminated illicit drugs
Bone meal calcium supplements
Lead plumbing
Folk remedies

TABLE II

SOURCES OF EXPOSURE TO LEAD OUTSIDE THE HOME

Dust, soil contamination
Burning batteries
Burning old painted wood
Lead gasoline, road dust
Old home renovation
Lead dust from work on clothes

TABLE III

SOURCES OF EXPOSURE TO LEAD INDUSTRIAL - WORKPLACE

Renovation, construction
Radiator, auto repair
Smelting operations
Soldering, welding
Battery manufacture
Painting, demolition
Firing ranges (indoor)

Contamination of water continues to be a problem in 1990[9,10]. Lead is leached from solder around joints of copper pipes, and in very old buildings from lead plumbing. Acid or hot water can increase the solubility of lead. Corrosion of lead pipes is often reduced over time due to the gradual deposition of phosphates that provide a protective film. The U.S. EPA in 1974 set a level of 50 µg per liter as the Maximum Contamination Level (MCL) allowable in public water systems. This is currently being considered for a reduction to 20 µg per liter.

The combustion of leaded gasoline has been the major source of lead in the air in urban communities since its introduction in 1925. It is estimated that over 90% of the air burden of lead is from this source[11]. Lead released into the air near roadways may settle onto soils and ducts. The association between high traffic density and high lead-levels in infants has been demonstrated[12]. The flow of dispersal of gasoline-based lead from combustion, to air, to food, to soil and into man has been shown[13]. The introduction of lead-free gasoline has contributed to a considerable decrease in the overall burden of lead in the air, and a reduction in mean blood lead-levels over the past 15 years[14].

Soil and dust are of particular concern in terms of possible sources of lead because of the tendency for children in the 2- to 3-year-old age group to put their hands, and other objects that may be contaminated with lead-containing dust, into their mouths. The contribution to the total lead intake in children by the ingestion of food and soil or dust is estimated to be approximately 67% and 20%, respectively (NAS, 1980). However, children have a greater risk of exposure to lead from sources other than air, food, or water, ie normal mouthing activity or abnormal ingestion of non-food items (pica).

Exposure to lead in the industrial workplace is the main source of toxicity in the case of adults. There is a list of 120 occupations that may be associated with a risk of undue exposure to lead[15]. In 1989 alone, the literature has documented lead poisoning in bridge-demolition workers[16], paint removal workers[17], battery-repair-shop workers[18], automobile radiator mechanics[19] and construction workers[20]. Workers in high-risk occupations have been shown to bring lead dust home to their families on contaminated clothing[21]. This means the physician has a responsibility to recognize the lead-exposed worker and to understand workplace standards for lead levels established by the federal Occupational Safety and Health Administration (OSHA)[22].

Recent concern over contamination of drinking water in South Kona caused environmental health officials to inspect homes where high levels of lead were found in rain catchment water. In addition to contamination by lead of the water catchment systems, inspectors found a variety of other sources of lead in areas where children could be potentially exposed.

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These included: Peeling, old, lead-based paint, old car batteries that were contaminating soil in the yards, lead fishing weights, leaded gasoline, lead shot, lead-lined art work, and lead cooking utensils. Fishing weights are often manufactured in the home, and if ventilation is inadequate, may be a source of exposure.

Pharmacokinetics of lead

> Lead is not known to have any useful biological purpose in the body. Its only value might be considered as a "marker" of environmental pollution. Ideally, a normal serum lead level should be zero.

Lead enters the body primarily through the gastrointestinal tract. An adult absorbs approximately 5% of a lead dose, and small infants and children may absorb as much as 20%. If there is a deficiency of iron, zinc, or calcium, absorption of lead may be increased[24].

The distribution of lead in the body is complex and poorly studied in man. The total body lead burden can be divided into 3 compartments with blood and soft tissue representing a rapidly interchangeable pool, the skeletal system as a storage pool, and skin and muscle as an intermediate pool. Ninety percent of the body burden of lead can be found in bone. It is attracted to the metaphyses of growing bones and can slow linear growth in children, therefore. When it is incorporated into cortical bone, it becomes an inert and insoluble lead phosphate. An equilibrium develops between blood lead and the insoluble bone lead in growing bone[25].

The elimination half-life of lead is difficult to assess due to the movement between compartments. The half-life of lead after a single toxic exposure is 27 to 44 days, whereas bone storage may extend the half-life in chronic exposure to 6 to 7 years[25,26]. The kidney is responsible for approximately 75% of the daily lead loss; feces, skin, and hair provide other routes of excretion.

Pathophysiology

Lead has a high affinity for negatively charged sulfhydryl groups found in enzyme systems throughout the body. One of the enzyme systems most sensitive to the presence of lead is the mechanism in heme biosynthesis. Disordered porphyrin metabolism and heme biosynthesis are affected at several enzymatic steps. When an enzyme pathway is blocked, there is an increased accumulation on one side of the equation and a decreased formation of the substance on the other side of the equation. When gamma aminolevulinic acid dehydratase and ferrochelatase are inhibited by lead, heme synthesis is reduced and anemia results[27]. However free erythrocyte protophorphyrin (FEP) accumulates on the other side of the equation. This is the basis for testing for lead poisoning.

Divalent lead is similar in many ways to calcium and exerts a competitive action on several systems, such as mitochondrial respiration, as well as on various neurological functions. The similarity between calcium and lead may explain partially why they seem interchangeable in bone; it may also account for the fact that 90% or more of the total body burden of lead is stored in the skeleton.

The effect of lead on mitochondrial structure and function is the main cause of central and peripheral nervous system toxicity. Lead is filtered through the blood-brain barrier more readily in children, causing a swelling and distortion of mitochondriae in the brain, which leads to coma, convulsions and cerebral edema. Peripheral nerves, on the other hand, can undergo segmental demyelination, with decreased motor nerve conduction velocity. Lead also appears to interfere with neurotransmitters. Aminolevulinic acid (ALA), which is produced as a result of action of lead on heme synthesis is chemically similar to the neurotransmitter gamma amino-isobutyric acid (GABA). ALA formed as a result of lead poisoning may bind to GABA sites producing neurotoxicity[28].

Lead affects the kidney adversely primarily in the proximal tubules and loops of Henle. A reversible acute renal failure with a Fanconi-like syndrome (renal tubular acidosis, glycosuria, and aminoaciduria) is seen in acute lead poisoning. Azotemia and hypertension can occur in chronic lead poisoning, as secondary to chronic interstitial changes[29]. Gout may occur as a result of diminished clearance of water, with subsequent hyperuricemia.

Clinical manifestations

Signs and symptoms of lead poisoning are dependent on the age of the patient, the duration of exposure and the lead level. Physical findings are confined to the hematologic, renal, nervous, gastrointestinal and cardiac systems (Table IV).

There are 2 clinically distinct syndromes associated with lead-poisoning. The acute clinical picture characterized by hepatic injury, hemolysis, and encephalopathy may occur when a large amount of lead is ingested and absorbed. Most lead-poisoning however is chronic, of slow onset, and is the result of the accumulation of lead in the body over time.

It is important to note that serious damage may occur in the absence of any signs or symptoms. Detection of lead-poisoning is best done by screening of the populations known to be at risk, such as children and pregnant women living in urban, pre-1950 housing, or workers in the lead industry. "At-risk" individuals can be recognized only by an alert and aware physician taking a careful work, environmental and medical history. If there is any possibility of exposure in a home, play or work environment, screening tests should be performed.

Hematologic signs and symptoms are the result of disordered heme synthesis and a resultant anemia. Significant anemia is a later manifestation of lead-poisoning; the absence of anemia does not rule out toxicity. Lead-related anemia might be normocytic and normochromic, without the presence of concomitant iron-deficiency anemia. Typical childhood lead-poisoning, however, usually results in a microcytic and hypochromic anemia associated with iron deficiency; the latter often potentiates the anemia of lead-poisoning. Basophilic stippling is a result of clumped endoplasmic reticulum[30]. The absence of basophilic stippling, however, does not rule out toxicity.

Renal symptoms are related to interstitial nephritis and tubular disturbances. Deposits of lead in the kidneys may produce glycosuria and aminoaciduria.

Gastrointestinal symptoms include some of the most common and frequent findings in lead-poisoning. They include nausea, vomiting, colicky abdominal pain, and constipation[31]. During symptoms of "lead colic," the blood pressure may be elevated and the pulse slow. The direct action of lead on smooth muscle has been suggested by some as the origin of colic, whereas others are of the opinion that vagal irritation associated with intestinal ischemia is the underlying mechanism. Hepatic injury occurs only in acute lead-intoxication.

Central and Peripheral Nervous Systems are affected by lead. The "wrist drop" or focal palsy is one of the earliest recognized signs of lead-poisoning. Peripheral neuropathy does not always correlate with blood levels or the duration of lead exposure[32]. Although increased lead-levels are known to cause slowing of nerve conduction velocity, it is not a useful test for low-level lead-toxicity[33]. Lead-encephalopathy, manifested by depression of the sensorium, increased intracranial pressure, vomiting, irritability, convulsions and coma, results in serious long-term effects. More subtle CNS findings include irritability, lethargy, fatigue, and headache.

Neurobehavioral affect in low-level exposure to lead has been studied and debated for over a decade. Needleman et al[34] demonstrated that asymptomatic, early, school-aged children with dentin lead-levels > 20 μg% had problems with classroom performance. These children were subsequently shown to have lower IQ scores by the 5th grade, and a higher risk of high school drop-out and absentee rates. A summary meta-analysis of modern studies of low-level, childhood exposure to lead strongly supports the hypothesis that even at low doses lead impairs children's IQ[35]. Children with even low levels of lead in the blood have demonstrated a lower vocabulary capacity, poor eye-hand coordination, prolonged reaction times, and decreased grammatical-reasoning scores. Abnormal blood lead-levels have also been associated with hearing deficits[36]. It is becoming increasingly more evident that exposure to lead during early development (fetus and young child) is a particularly serious health problem in terms of central nervous system development as the target organ for toxicity. Delayed cognition, decreased IQ and hearing impairment can occur at lead-levels previously thought to cause no harm[37]. Cord blood lead-levels have shown that about 80% of the maternal blood lead goes to the developing fetus[38]. Developmental delays have been demonstrated in children born with blood lead-levels > 10 μg%[39].

TABLE IV

SIGNS AND SYMPTOMS OF LEAD-POISONING

HEMATOLOGIC	GASTROINTESTINAL
Anemia (Microcytic)	Abdominal pain
Hemolytic anemia	Constipation
Basophilic stippling	Anorexia
	Nausea, vomiting
	"Metallic" taste
RENAL	NEUROLOGICAL
Proteinuria, glycosuria	Encephalopathy
Aminoaciduria	Developmental delays
Interstitial nephritis	Peripheral neuropathy
Decreased GFR	Mental deficiency
Hypertension	Hearing loss

Laboratory assessment and screening

Because the symptoms of lead-poisoning can be so subtle and insidious in onset, appropriate laboratory screening of high-risk groups becomes very important. An elevated blood lead-level is necessary for the diagnosis of lead-poisoning and also in monitoring subsequent appropriate therapy. However, blood lead-level testing is expensive, easily contaminated and very difficult to control. It is not practical for mass screening.

Analysis of the accumulation products of disordered heme synthesis is a more useful way for mass screening. Erythrocyte protoporphyrin can be easily measured in a wet drop of blood in a hematofluorometer[40]. "Free" erythrocyte protoporphyrin (FEP), zinc protoporphyrin (ZPP), and EP are essentially equivalent measurements; ZPP or FEP in the screening of cohorts of high-risk groups are the most cost-effective tests available. They will test for both lead poisoning and iron deficiency at the same time[37], a value > 35 μg/dl indicates the presence of lead. Significant lead-poisoning will usually be indicated by ZPP levels > 100 μg/dl. In children with normal iron status, however, ZPP may not be a sensitive indicator of blood lead-levels[41], and may be less effective in identifying children with a low lead-burden. The federal Center for Disease Control (CDC) has developed guidelines for interpretation of ZPP results[42]. Table 5 summarizes CDC recommendations, providing a plan for diagnostic and therapeutic decisions based on ZPP and blood lead-levels. There should be a greater urgency for evaluation if the patient is symptomatic, if under 3-years-of-age, or if other family members show evidence of lead-toxicity.

The American Academy of Pediatrics Committee on Environmental and Health Hazards has proposed several specific recommendations for both practitioners and public agencies[37]. The recommendations are based on the fact that exposure to lead is widespread and causes serious and generally irreversible neuropsychological effects at even low exposure levels. Recommendations to private practitioners include more widespread use of the FEP test as a sensitive indicator of subclinical iron-deficiency, as well as screening for lead. Particular emphasis should be directed towards screening children under 3-years-of-age who live in or frequent high-risk environments. The Academy further recommends that pediatricians take responsibility for educating parents of children living in high-risk areas about the hazards of lead and how to prevent exposure to it.

Recommendations to public agencies by the Academy include mandatory reporting of all cases of lead-poisoning to the state health departments, the development of national programs for the screening for hazards in housing, and for the systematic decontamination of houses that still contain lead-based paint. These recommendations by the Academy for an organized effort to study, identify and reduce the hazards of contamination in our environment by lead has resulted in Congressional action and a comprehensive review of the problem this past year[14].

Therapy

"Urine provocation testing" is used to determine the need

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for chelation therapy. An asymptomatic patient who fits into a grade II or III toxicity range (Table V) should have provocative testing with $CaNa_2EDTA$ to demonstrate the amount of mobilizable lead. One dose of 25 mg/kg in 0.5% solution should be given over one hour[43]. Urine is then collected for 8 hours to determine the lead excretion rate. If greater than 0.6 µg of lead is excreted per mg EDTA administered, the test is positive and further chelation therapy is indicated (Table 6). $CaNa_2EDTA$ is a chelating agent that removes lead from the "extracellular" compartment. It draws lead from soft tissue, from the central nervous system and from red blood cells. It is equally effective given i.v. or i.m. in a daily dose of 50-75 mg/kg. Since EDTA will chelate other inherent metals in the body such as zinc, it should not be given for more than 5 days without an interval of 2 days.

In the instance of severe toxicity, dimercaprol (BAL) should be given prior to the use of EDTA. BAL will enter cells and tie up available lead avoiding a rapid increase in serum lead levels secondary to mobilization by EDTA[44]. EDTA-lead complexes can be very nephrotoxic; therefore it is important to maintain good renal output during chelation therapy. Table 6 provides guidelines for chelation therapy based on the clinical state and laboratory values.

TABLE V

ZINC PROTOPHORPHYRIN BY HEMATOFLUOROMETER: RISK CLASSIFICATION OF ASYMPTOMATIC CHILDREN FOR PRIORITY MEDICAL EVALUATION

BLOOD LEAD ug/dl	ERYTHROCYTE PROTOPORPHYRIN ug/dl			
	<35	35-74	75-174	>175
Not Done	I	*	*	*
<24	I	↓Fe	↓Fe	EPP
25-49	A	II	III	III
50-69	?	III	III	IV
<70	?	?	IV	IV

* = Blood test needed to estimate risk
↓Fe = Iron deficiency anemia
A = Early exposure or contamination
? = Combination not generally observed, repeat analysis
EPP = Consider erhythrocytic protoporphyrin
I = Normal
II = Moderate toxicity, limit exposure
III = Severe toxicity, assess early
IV = Urgent Toxicity, hospitalize

(Adapted from CDC Guidelines42)

TABLE VI

THERAPY OF LEAD POISONING

CLINICAL STATE	THERAPY
1) Blood lead 25-50 ug/dl Urine provocation < 0.6 ug/Pb < 0.6 mg EDTA	1) -Remove source of lead -Close follow-up
2) Blood lead < 50 ug/dl Urine provocation < 0.6	2) -EDTA 50 mg/kg x 5 days -Close follow-up
3) Blood lead 50-69 ug/dl	3) -Assure urine output -EDTA 50 mg/kg x 5 days -Monitor rebound in 5-7 days -Repeat if necessary
4) Blood lead < 70 ug/dl	4) -BAL 3 mg/kg q f h x 2 days -then q 4-6 h x 2 days -then q 4-12 h up to 7 days -(discontinue when lead < 50 ug/dl)
5) Lead encephalopathy	5) -BAL 5 mg/kg/dose as in #4 -EDTA 75 mg/kg/day -Continue for 5 days, then rest 2 days and repeat

(Modified from Piomelli et al45)

The practicing physician and prevention

In the absence of smelters, foundries, high-risk work environments, the decrease in the use of leaded gasoline, and the relatively small number of pre-1950 housing, Hawaii is a "low-risk" environment for lead-poisoning.

However, exposure to lead is widespread and in unexpected forms in our environment. It is quite clear that even minimum but chronic exposure to lead can result in neuropsychologic effects that are often irreversible. Prevention of exposure to lead is far more worthwhile than the recognition and then treatment of the signs and symptoms once they have occurred.

There are no "safe" levels of lead in the environment. Recent trends in prevention stress the need to reduce exposure to lead to the greatest extent possible. Since only a relatively small fraction of all children can be effectively protected by the identification and removal of hazards, a more effective public health control involving the removal of sources of lead that may contaminate soil, dust, air, water and food, is needed.

Methods are available and have been shown to be effective in reducing exposure to lead. For example, the rigorous practices of hygiene has been found to be effective in reducing the likelihood of elevated blood lead in children (Charney, 1983). Such may include the frequent mopping of floors, tabletops and shelves and other surfaces upon which particles may accumulate as well as frequent washing of hands, especially before meals.

The physician must maintain a keen index of suspicion when high-risk populations and high-risk environments suggest an exposure to lead. Anyone at high risk should be screened for the presence of lead whether or not signs or symptoms exist.

Lead-poisoning must, by law, be reported to the DoH. Through surveillance by the authorities, high-risk populations and high-risk environments can be further identified in Hawaii.

BIBLIOGRAPHY

1. Waldron HA. Chasing the lead. *Br Med* J 1985; 291:366-367.
2. Chisolm JJ Jr. Ancient sources of lead and lead poisoning in the United States today. *West J Med* 1985;143:380-381.
3. Bose A, Vashitha K, O'loughlin BJ. Azarcon por empacho; another cause

of lead toxicity. *Pediatrics* 1983;72:106-108.
4. McCord CP. Lead and lead poisoning in early America: Benjamin Franklin and lead poisoning. *Ind Med Sur* 1953;22:394.
5. Ellenhorn MU, Barceloux DG. Medical Toxicology. Elsevier Science Publishing Co., New York, 1988, pp 1030-1041.
6. Rhoads F. Personal communication. Unpublished Data.
7. Joselow MM, Banth JE, Fisher W, Valentine J. Environmental contrasts: Blood lead-levels of children in Honolulu and Newark. *J Environ Health* 1974;37:10-12.
8. Curran JP, Nunez JR. Lead poisoning during home renovation. *NY State J Med* 1989;89:679-680.
9. Jones RR. The continuing hazard of lead in drinking water. *Lancet* 1989;2:669-670.
10. Marcus WL. Lead health effects in drinking water. *Toxicol and Indust Health* 1986;2:363-407.
11. Fergusson J. Lead: petrol lead in the environment and its contribution to human blood levels. *Sci Tot Environ* 1986;50:1-54.
12. Lyngbye T, Hansen O, Grandjean P, Trillingsgaard A, Beese I. Traffic as a source of lead exposure in childhood. *Sci Tot Environ* 1988;71:461-467.
13. World Health Organization. Regional office for Europe: air quality guidelines, 1986; (Review draft, WHO vol. II. Lead, Ch. 19, 1-34.)
14. Agency for Toxic Substances and Disease Registry. The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: US Department of Health and Human Services, 1988; DHHS document No. 99-2966.
15. Environmental Studies Board. Lead in the human environment: a report prepared by the Committee of lead in the human environment. Washington, D.C.: Commission on Natural Resources, National Research Council, National Academy of Sciences, 1980.
16. Himmelstein J, Wolfson, M, Pransky, G, Morse D, Ross A, Gill J. Lead Poisoning in bridge demolition workers - Massachusetts. *JAMA* 1989; 262:2369-2370.
17. Christophers AJ. Lead intoxication in paint removal workers. *Med J Australia* 1987;146:599-560.
18. Figueroa JP, Keenlyside RA. Occupational and environmental lead poisoning associated with battery repair shops - Jamaica. *MMWR* 1989; 38:474, 479-481.
19. Goldman RH, Baker EL, Hannan M, Kamerow DB. Lead poisoning in automobile repair mechanics. *N Engl J Med* 1987;317:214-218.
20. Marino P, Franzblau A, Lilis R, Landrigan P. Acute lead poisoning in construction workers: the failure of current protective standards. *Arch Environ Health* 1989;44:140-145.
21. Johnson D, Houghton K, Siegel C, Martyny J, Mangione EJ. Occupational and paraoccupational exposure to lead - Colorado. *MMWR* 1989;38:338, 340-345.
22. Rempel D. The lead-exposed worker. *JAMA* 1989;262:532-534.
23. Mahaffey KR, Annest JL, Roberts J, et al (Food and Drug Admin Cincinnati, OH, USA) national estimates of blood levels: demographic and socioeconimic factors. *N Engl J Med* 1982;307:573-579.
24. Mahaffey KR. Nutritional factors in lead poisoning. *Nutr Rev* 1981; 39:353-365.
25. Marcus AH. Multicompartment kinetic modules for lead. Linear kinetics and variable absorption in humans without excessive lead exposure. *Environ Res* 1985;36;459-472.
26. Schutz A, Skerfving S, Ranstam J, et al. Kinetics of lead in blood after the end of occupational exposure. *Scand J Work Environ Health* 1987;13:221-231.
27. Ringeberg QS, Doll D, Patterson WP, Perry M, Yarbro J. Hematologic effects of heavy metal poisoning. *So Med J* 1988;81:1132-1139.
28. Clarkson T. Metal toxicity in the central nervous system. *Environ Health Pers* 1987;75:59-64.
29. Wedeen R, D'Haese P, Van de Vyver F, Verpooten G, De Broe M. Lead nephropathy. *Amer J Kidney Dis* 1986;5:380-383.
30. Angle Cr, McIntyre MS. Low level lead and inhibition of erythrocyte pyrimidine nucleotidase. *Environ Res* 1978;17:296-302.
31. Dahlgren J. Abdominal pain in lead workers. *Arch Environ Health* 1978;133:156-159.
32. Ehle AL. Lead neuropathy and electrophysiological studies in low level lead exposure: a critical review. *Neurotoxicology* 1986;7:203-216.
33. Schwartz J, Landrigan P, Feldman R, Silbergeld E, Baker E, Von Lindern I. Threshold effect in lead-induced peripheral neuropathy. *J Pediatrics* 1988; 112:12-17.
34. Needleman H, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance in children with elevated dentine lead levels. *New Engl J Med* 1979;31:258-264.
35. Needleman H, Gatsonis A. Low level lead exposure and the IQ of children: a meta-analysis of mordern studies. *JAMA* 1990;263:673-678.
36. Schwartz J, Otto D. Blood lead, hearing thresholds, and neurobehavioral development in children and youth. *Arch Environ Health* 1987;42:153-160.
37. Committee on Environmental Hazards, Committee on Accident and Poison Prevention. Statement on Childhood Lead Poisoning; *Pediatrics* 1987; 79(3):457-465.
38. Bellinger D, Leviton A, Rabinowitz M, et al. Correlates of low-level lead exposure in urban children at 2 years ofd age. *Pediatrics* 1986;77:826-833.
39. Bellinger D, Leviton A, Waternaux C, et al. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med* 1987;316:1037-1043.
40. Blumberg WE, Eisinger J, Lamola AA, et al. Zinc protoporphyrin level in blood determined by a portable hemtofluorometer: a screening device for lead poisoning. J *Lab Clin Med* 1978;89(4):712.
41. Clark M, Royal J, Seeler R: interaction of iron deficiency and lead and the hematologic findings in children with severe lead poisoning. *Pediatrics* 1988; 81:247-254.
42. Centers for Disease Control. Preventing lead poisoning in young children. U.S. Department of Health and Human Services/Public Health Services, January 1985.
43. Needleman H. The persistent threat of lead: Medical and sociological issues. In Current Problems in Pediatrics, Vol XVIII, No 12, Jean D. Lockhart (ed). Yearbook Medical Publishers, Inc., December 1988.
44. Chisholm JJ Jr: Treatment of acute lead intoxication - choice of chelating agents and supportive therapeutic measures. *Clin Toxicol* 1970;3:527.
45. Piomelli S, Rosen JR, Chisolm J Jr., et al. (Columbia Univ, Coll Physicians & Surgeons, New York, NY, USA). Management of childhood lead poisoning. (Review Article) *J Pediatr* 1984;105:523-532.

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